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Arterial stiffness and its relationship to clinic and ambulatory blood pressure: a longitudinal study in non-dialysis chronic kidney disease

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ABSTRACT

Background. Both arterial stiffness and systolic blood pressure (BP) are established cardiovascular risk factors, yet little is known about their interrelationship in chronic kidney disease (CKD). The goal of this prospective study was to describe the trajectory of aortic pulse wave velocity (PWV) and BP and to compare the longitudinal interrelationship of BP (clinic and 24 h ambulatory recording) with the PWV.

Methods. Clinic BP was taken in two ways: at the time of the measurement of the PWV (Clinic-S) and as an average of triplicate measurements on three separate occasions within 1 week (Clinic-M). 24 h ambulatory BP was measured using a validated monitor and PWV was measured in the aorta using an echo-Doppler technique.

Results. Among 255 veterans with CKD followed for over up to 4 years, the rate of change of log PWV was inversely related to the baseline PWV; the trajectories were variable among individuals and the net population change was no different from zero. In contrast, systolic BP significantly increased, but linearly, and a strong relationship was seen between cross-sectional and longitudinal changes in Clinic-M systolic BP and log PWV. In contrast, a longitudinal relationship between Clinic-S and log PWV was absent. In the case of 24-h ambulatory BP, a strong cross-sectional change was seen between awake and 24 h systolic BP but not between sleep BP and log PWV

Conclusion. In conclusion, among people with CKD, the PWV changes over time and is inversely related to the baseline PWV. An average of clinic BP measurements taken over three visits, but not single measurements, are useful to assess the PWV and its change over time. Differences exist between ambulatory BP monitoring recording during the sleep and awake states in their ability to predict the PWV. Taken together, these data support the view that among those with CKD not on dialysis, targeting clinic BP taken on multiple occasions using a standardized methodology or daytime ambulatory systolic BP may slow the progression of arterial damage.

Keywords: ambulatory blood pressure monitoring, arterial stiffness, blood pressure, chronic renal failure, pulse wave velocity

INTRODUCTION

Both hypertension and chronic kidney disease (CKD) are common in the adult US population [1] and they together account for a large burden of cardiovascular morbidity, mortality and costs associated with CKD [2]. Among veterans, CKD rivals diabetes mellitus as a coronary risk equivalent [3]. Established risk factors for cardiovascular disease include elevated arterial stiffness and blood pressure (BP) [4]. Arterial stiffness is best measured using the aortic pulse wave velocity (PWV) [5].

Although both PWV and BP are powerful cardiovascular risk factors, little information is available on how PWV evolves in people with CKD not on dialysis. The cross-sectional relationship between PWV and BP is well recognized, but among those with CKD, how PWV changes over time and how BP predicts this change is poorly understood.

The understanding of which BP measurement method predicts target organ damage as assessed by direct measurement of PWV in the elastic aorta is important. This is because if one technique of BP measurement has a stronger relationship than the other it would suggest that targeting that BP would be of greater clinical importance. For example, although guidelines suggest using multiple occasions for the measurement of clinic BP prior to making clinical decisions, most clinical decisions are still made using a single measurement occasion, which is at the time of the clinic visit. Whether the ability to predict target organ damage is similar in those with a single clinic BP measurement versus those with multiple clinic measurements remains unknown. Furthermore, accumulating evidence suggests that BP measurements made outside the clinic may provide prognostically superior information [6–9]. However, the comparative value of BP obtained in the clinic and that obtained using 24-h ambulatory BP in assessing the PWV remains unclear.

In this study, the author explored the trajectory and pattern of change of the PWV, clinic systolic BP and 24-h ambulatory BP and compared the two types of clinic BP, measurement on a single occasion (Clinic-S) and measurement on multiple occasions (Clinic-M), in their ability to predict cross-sectional PWV and its trajectory. Similarly, the author compared 24-h ambulatory BP measurement and its components, awake or sleep ambulatory BP, for their ability to predict cross-sectional PWV and its trajectory.

MATERIALS AND METHODS

Details of this cohort has been published previously [10]. Briefly, this was a prospective study of patients with CKD stages 2 through 4 [estimated glomerular filtration rate (GFR) defined using the MDRD equation <90 mL/min/1.73 m^2 but >15 mL/min/1.73 m^2]. For those with stage 2 CKD, albuminuria (A2 or >300 mg/g creatinine) was required for inclusion in the cohort. Those with an initial clinic BP of $\leq$140/90 mmHg were considered eligible and studied further. However, $\sim$10% of people with a single clinic BP of $\leq$140/90 mmHg were found to be hypertensive on further evaluation and were not excluded.

After obtaining a clinical history, performing a physical examination and obtaining basic laboratory tests, measurements of BP in the clinic [average of three visits (Clinic-M)] and by 24-h ambulatory monitoring (24 h average) were performed as reported earlier. BP measurements in triplicate were also obtained independent of the three clinic visits at the time of the PWV measurement. BP obtained during the PWV measurement was called the single-visit clinic BP (Clinic-S).

Classification of hypertension

The original definition of masked hypertension proposed by Pickering *et al.* [11] and the one used by the International Database on Ambulatory Blood Pressure in Relation to Cardiovascular Outcomes (IDACO) investigators [12] do not take into account BP recordings at night. Accordingly, masked uncontrolled hypertension was defined as controlled clinic BP ($<$140/90 mmHg on average of three clinic visits by oscillometric BP measurement) but elevated ambulatory BP [13]. Elevated ambulatory BP was defined as elevated daytime ($\geq$135/85 mmHg) BP. To define daytime and nighttime, we used patient diaries. Elevated ambulatory BP was also defined in two other ways: (i) elevated 24-h ($\geq$130/80 mmHg); and (ii) either elevated daytime or elevated nighttime ($\geq$120/70 mmHg) ambulatory BP.

PWV measurements

Arterial stiffness was assessed by measuring the aortic PWV through direct visualization of the descending aorta with the use of an echo-Doppler technique (Acuson Cypress, Seimens Medical). The flow pulse was recorded by continuous Doppler from the root of the left subclavian artery and just proximal to the bifurcation of the abdominal aorta with simultaneous electrocardiographic recording [14]. The length of the descending aorta was estimated by measuring the body surface distance from the suprasternal notch to the recording site of the aortic signal (near the umbilicus). The time elapsed from the peak of the R wave to the foot of the systolic impulse was recorded over six beats. The length of the descending aorta divided by the difference between transit times was calculated to yield the aortic PWV [14].

Statistical analysis

Line graphs of individual level data were generated and relationships of slopes and intercepts plotted prior to model fitting. Linear mixed models were then used to assess the trajectory of the PWV and BP over time [15]. A random coefficient model was fitted [16]. BP was modeled separately for each measurement type (Clinic-S, Clinic-M, 24 h ambulatory, awake ambulatory, sleep ambulatory).

Because all BP measurements were obtained in the same participants, the beta coefficients for PWV change were not multiply adjusted for differences in confounders. This was because the question of what the independent predictors of PWV change are was not being asked; the BP values were simply being compared as to their relative ability to predict PWV change. When comparing different BP measurements and outcomes, a similar approach has been adopted by us and others [17, 18].

All statistical analyses were done with Stata 14.0 (Stata Corp, College Station, TX, USA). The nominal level of statistical significance was taken as a two-sided P of 0.05.

RESULTS

Baseline characteristics of participants are given in Table 1. As expected of a veteran population, participants were older, mostly White men, and two-thirds were past smokers. Notable was a high prevalence of diabetes mellitus and cardiovascular disease ascertained by a review of medical records. The average estimated GFR was 44 mL/min/1.73 m^2 and the median urinary

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Table 1. Baseline characteristics of the study sample

	Overall	Group with >1 PWV	PWV decreased	PWV increased	P-value
Number of participants, *n* (%)	255	66	28 (42.4%)	38 (57.5%)	
Age (years)	69.4 ± 9.9	67.4 ± 9.9	66.7 ± 11.5	67.9 ± 8.6	>0.2
Male sex, *n* (%)	249 (97.6%)	65 (98.4%)	27 (96.4%)	38 (100%)	>0.2
Race, *n* (%)					
White	201 (78.8%)	50 (75.7%)	21 (75%)	29 (76.3%)	>0.2
Black	47 (18.4%)	16 (24.2%)	7 (25%)	9 (23.6%)	
Other	7 (2.74%)				
Height (in)	174.7 ± 7.8	174.6 ± 7.4	173.2 ± 7.1	175.7 ± 7.6	0.18
Weight (kg)	94.2 ± 16.6	93.3 ± 16.3	93.8 ± 15.6	93 ± 17	>0.2
Body mass index (kg/m^2)	30.8 ± 4.6	30.5 ± 4.4	31.2 ± 4.4	30 ± 4.4	>0.2
Diabetes mellitus, *n* (%)	167 (65.4%)	45 (68.1%)	20 (71.4%)	25 (65.7%)	>0.2
Myocardial infarction, *n* (%)	74 (29.0%)	23 (34.8%)	7 (25%)	16 (42.1%)	0.15
Coronary artery bypass graft, *n* (%)	55 (21.5%)	22 (33.3%)	8 (28.5%)	14 (36.8%)	>0.2
Congestive heart failure, *n* (%)	48 (18.8%)	15 (22.7%)	4 (14.2%)	11 (28.9%)	0.16
Peripheral vascular disease, *n* (%)	53 (20.7%)	13 (19.6%)	7 (25%)	6 (15.7%)	>0.2
Smoking, *n* (%)					
Never	42 (16.4%)	9 (13.6%)	4 (14.2%)	5 (13.1%)	>0.2
Past	175 (68.6%)	45 (68.1%)	18 (64.2%)	27 (71.0%)	
Current	38 (14.9%)	12 (18.1%)	6 (21.4%)	6 (15.7%)	
Laboratory tests					
Albumin (g/dL)	3.94 ± .47	4.05 ± .38	4.1 ± .45	4.02 ± .32	>0.2
Hemoglobin (g/dL)	13.2 ± 1.7	13.3 ± 2.1	13.2 ± 2.1	13.5 ± 2	>0.2
Estimated GFR (mL/min/1.73 m^2)	43.6 ± 15.6	46.7 ± 18.6	50.8 ± 23	43.7 ± 14	0.12
Log urine albumin/creatinine ratio	−3.24 ± 2.19	−3.36 ± 2.15	−3.03 ± 2.24	−3.61 ± 2.08	>0.2
Antihypertensive drugs (*n*)	3.1 ± 1.4	3.2 ± 1.6	3 ± 1.1	3.3 ± 1.8	>0.2
Angiotensin-converting enzyme inhibitors, *n* (%)	129 (51.3%)	37 (56.0%)	17 (60.7%)	20 (52.6%)	>0.2
Angiotensin receptor blockers, *n* (%)	56 (22.3%)	16 (24.2%)	7 (25%)	9 (23.6%)	>0.2
β-Blockers, *n* (%)	168 (66.9%)	42 (63.6%)	19 (67.8%)	23 (60.5%)	>0.2
Single-visit clinic systolic BP (mmHg)	135.2 ± 19.8	132.7 ± 16.2	133.1 ± 16.3	132.5 ± 16.3	>0.2
Single-visit clinic diastolic BP (mmHg)	72.3 ± 12.2	73.9 ± 10.9	73.1 ± 10.6	74.5 ± 11.3	>0.2
Single-visit clinic pulse rate (beats/min)	65 ± 11.7	63.5 ± 11.8	62.6 ± 12.2	64.1 ± 11.6	>0.2
Multiple-visit clinic systolic BP (mmHg)	122 ± 14.8	120.3 ± 14.1	120.9 ± 13.6	119.8 ± 14.6	>0.2
Multiple-visit clinic diastolic BP (mmHg)	60.8 ± 10.5	59.4 ± 10.5	58.2 ± 11	60.3 ± 10.1	>0.2
Multiple-visit clinic pulse rate (beats/min)	66.3 ± 11.7	64.1 ± 11.8	63.9 ± 13.4	64.3 ± 10.6	>0.2
24 h ambulatory systolic BP (mmHg)	128.4 ± 14.1	128.7 ± 14	127.7 ± 14.7	129.4 ± 13.6	>0.2
24 h ambulatory diastolic BP (mmHg)	69.9 ± 9.6	70.1 ± 8.9	68 ± 9.1	71.6 ± 8.6	0.11
24 h ambulatory pulse rate (beats/min)	68.8 ± 10.6	67.5 ± 11	67.3 ± 12.1	67.7 ± 10.2	>0.2
Sleep ambulatory systolic BP (mmHg)	124.6 ± 16.1	124.6 ± 14.8	122.4 ± 15.5	126.4 ± 14.3	>0.2
Sleep ambulatory diastolic BP (mmHg)	66.1 ± 10.4	66.6 ± 9.7	64.1 ± 9.2	68.5 ± 9.8	0.07
Sleep ambulatory pulse rate (beats/min)	65.6 ± 10.3	64.3 ± 10.2	63.3 ± 9.8	65.1 ± 10.6	>0.2
Awake ambulatory systolic BP (mmHg)	130.7 ± 14.1	131.1 ± 14.3	130.8 ± 14.8	131.3 ± 14	>0.2
Awake ambulatory diastolic BP (mmHg)	72.1 ± 9.8	72.3 ± 9.3	70.5 ± 10	73.6 ± 8.6	0.18
Awake ambulatory pulse rate (beats/min)	70.6 ± 11.3	69.5 ± 12.1	69.9 ± 13.9	69.2 ± 10.6	>0.2

Data are presented as mean ± SD or *n* (%). P-values reflect significance of differences between groups in which the PWV increased versus those in which the PWV decreased. Missing data on 1 for albumin, 2 for urine albumin and 2 for ambulatory in the overall sample.

albumin/creatinine ratio was 30 mg/g (interquartile range 8–280 mg/g). All but four participants were receiving antihypertensive drugs for BP control and the average number of antihypertensive drugs used was 3.1. At baseline, the Clinic-S measurement was 134/72 mmHg and the average Clinic-M was much lower at 121/60 mmHg. On average the 24-h ambulatory BP was 127/69 mmHg (123/66 mmHg during sleep and 130/72 mmHg during the awake state).

Participants who had more than one measurement of the PWV were well matched to the overall sample, as illustrated in Table 1.

Growth models of PWV and ambulatory systolic BP

Figure 1 shows the individual plots of each participant for the PWV, clinic and ambulatory BP measurements. The mean change is shown by the superimposed linear regression line. A linear change appears apparent for BP and no change for the PWV. Figure 2 shows the relationship between slopes and intercepts derived from each individual participant by ordinary least squares regression. Error bars of the slopes are shown where more than two measurements of the PWV or BP were available. Slopes of the PWV showed an inverse log–linear relationship with baseline PWV. Slopes of BP showed an inverse relationship with baseline BP in each case.

Table 2 shows the taxonomy of models for PWV, clinic and ambulatory BP measurements. In each case, the unconditional means model is described followed by the growth curve model. The unconditional means model accounts for the nested nature of the observations within participants. The natural log of PWV was taken as the outcome variable to normalize the data. The unconditional geometric mean PWV was 7.1 m/s (exponent of 1.96). The between-subject standard deviation (SD) of the log-

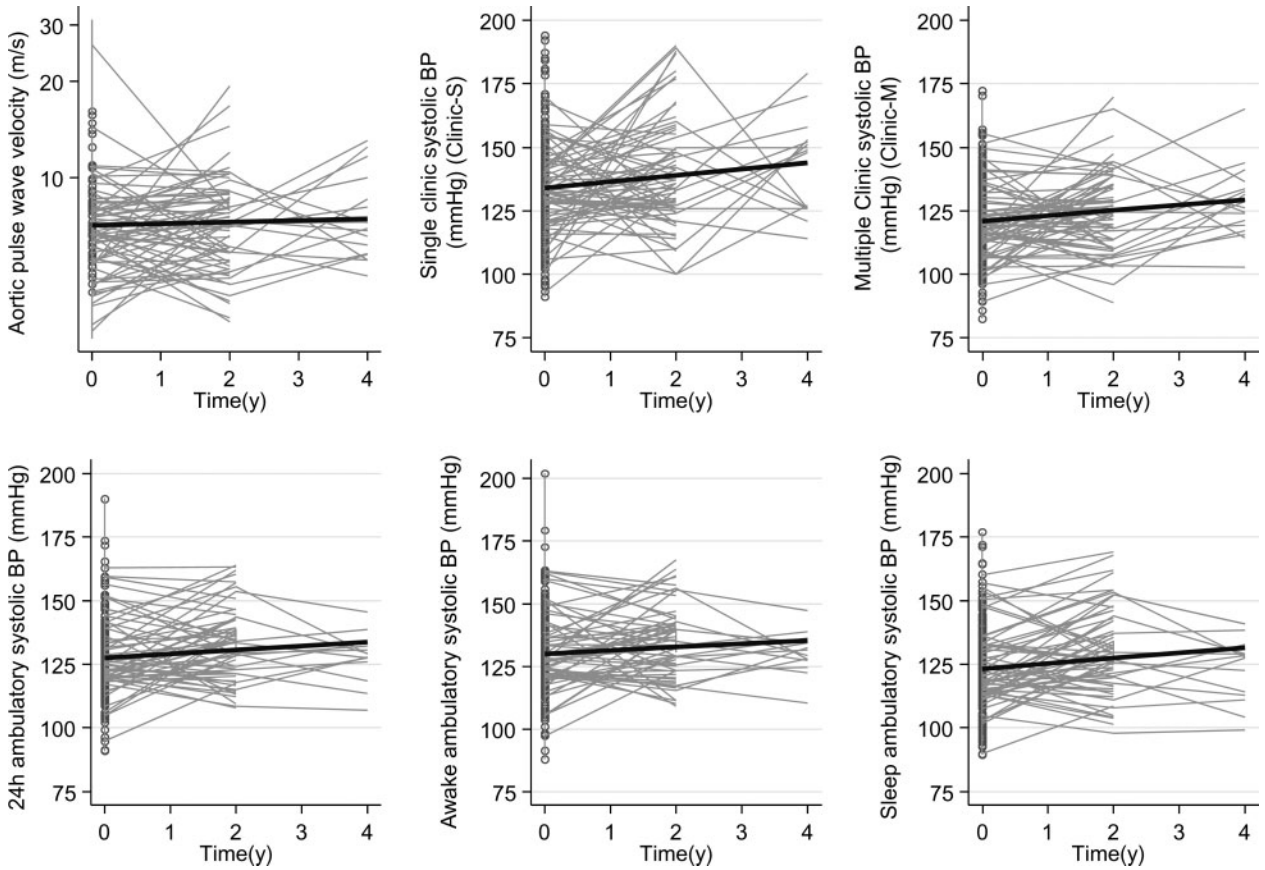

FIGURE 1: Trajectories of the PWV and BP over 4 years. Lines represent each individual participant. Circles at time zero are those who had a baseline measurement. Ambulatory BP monitoring was performed over 24 h. Clinic BP at a single visit (Clinic-S) and multiple visits (Clinic-M) are as described in the Materials and methods section.

transformed data may be interpreted as the coefficient of variation, or test–retest reliability, which was 16.3%. There was no overall change in the PWV with some participants showing an increment on the PWV, whereas others showed decrements. Table 1 gives the baseline characteristics of participants who have a PWV slope of <0 versus those who had a slope of >0. The two groups had no imbalance in any of the characteristics that could meaningfully change the slopes. Accordingly, there did not appear to be heterogeneity in the baseline characteristics that likely accounted for the variability in the slopes.

Single clinic systolic BP showed a large amount of variability within participants with intraclass correlation coefficient over years approaching zero. Multiple clinic BP values averaged 121.9 with a between-subject SD of 8.6 mmHg (intraclass correlation coefficient was 0.31). On average clinic BP (Clinic-M) increased by 2.1 mmHg/year. The magnitude of increase in 24-h ambulatory BP was less at 1.7 mmHg/year; however, the intraclass correlation coefficient was 0.41. Notably, the growth in sleep ambulatory BP was 2.3 mmHg/year, which was 64% more than that seen during the awake state. Furthermore, compared with the awake ambulatory systolic BP, numerically there was greater test–retest reliability for sleep ambulatory BP (intraclass correlation coefficient for awake BP being 0.32 and that for sleep BP being 0.50).

PWV and its relationship with clinic and ambulatory BP

The relationship of systolic BP growth and PWV was analyzed using mixed models and is summarized in Figure 3. In the case of single clinic BP measurement, every 10 mmHg increase in systolic BP across individuals was associated with a 3.34% increase in the PWV (this is shown as the intercept). Intra-individual increase of 10 mmHg in clinic systolic BP was associated with non-significant −0.51% growth in the PWV. In contrast, multiple clinic BP measurements were associated with a 4.69% greater PWV at baseline and 4.15% growth over time per 10 mmHg increment in systolic BP. These results were significant as noted by the P-values and the 95% confidence intervals not crossing 0. In comparison, for 24-h ambulatory BP [3.6% intercept, 2.3% (not significant, NS) slope], cross-sectional associations were positive and longitudinal associations were not. Furthermore, daytime ambulatory systolic BP [3.6% intercept, 2.8% slope (NS)] and sleep ambulatory BP [2.4% intercept (NS) 0.8% slope (NS)] show greater predictive power in the awake than in the sleep state (likelihood ratio test of nested models P = 0.027).

DISCUSSION

Although CKD is said to be a state of accelerated vascular ageing, among 255 veterans with CKD, over the 2–4 years of follow-up, we found little change in the mean PWV. Thus, secular increase in the PWV is not a fact of ageing in CKD; this is consistent with several reports among hemodialysis patients. Guerin *et al.* over a mean follow-up of 51 months reported a

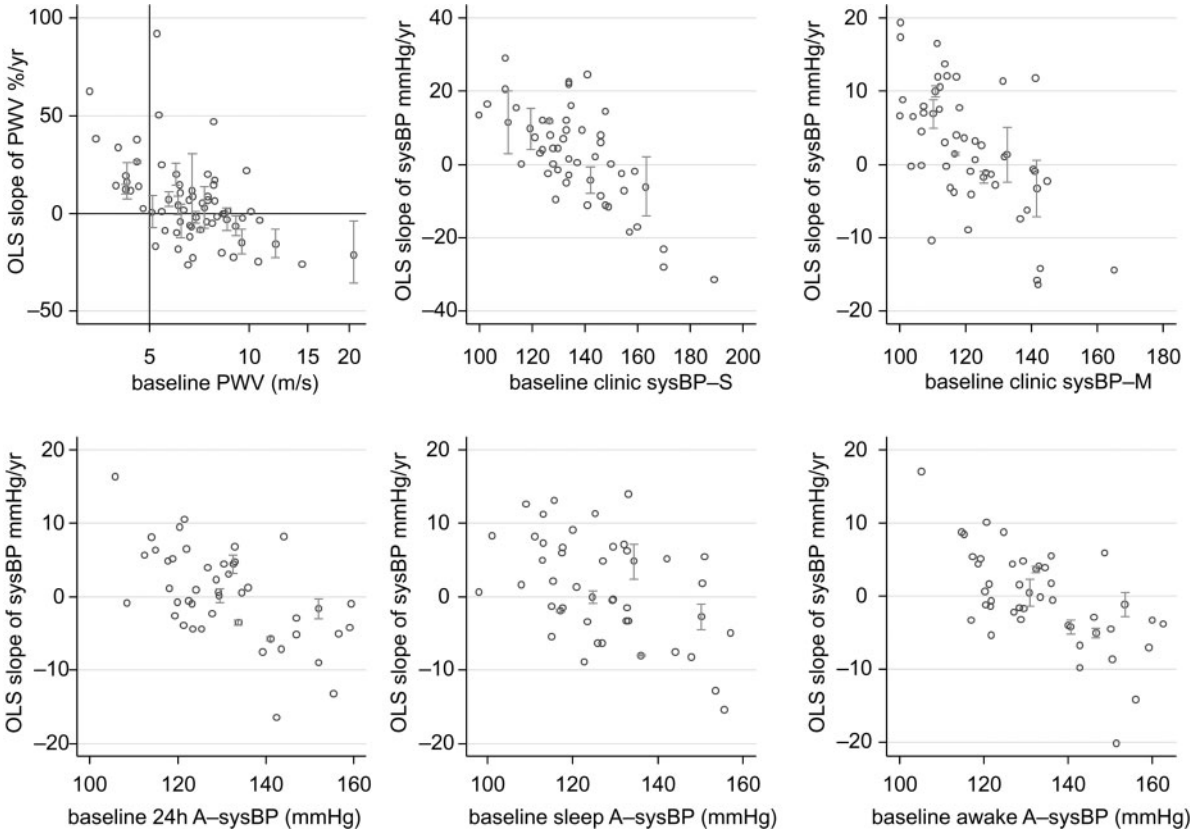

FIGURE 2: Relationship of ordinary least square (OLS) slopes and intercepts for individual participants. Error bars represent the standard error of the estimate in those participants who had three measurements. Baseline PWV is plotted on a log scale. All participants who had baseline PWV of <5 m/s had growth in PWV, whereas some with a PWV >5 m/s at baseline had regression of PWV. A-sysBP = ambulatory systolic BP.

Table 2. Taxonomy of models for the PWV and systolic ambulatory BPs

Parameter	Intercept	Slope	P for slope	σ_0	σ_1	σ_{01}	σ_e
Natural log of PWV (m/s)		log units/year					
Unconditional means only	1.960 (1.921–1.999)			0.163			0.302
Unconditional slope model	1.954 (1.911–1.996)	0.015 (−0.019–0.049)	0.39	0.2	0.052	−0.56	0.284
Single clinic systolic BP (mmHg)		mmHg/year					
Unconditional means only	135.4 (133.2–137.5)			0.00			20.00
Unconditional slope model	133.9 (131.6–136.3)	2.8 (0.5–5.1)	0.018	0.44	2.88	1.00	19.51
Multiple clinic systolic BP (mmHg)							
Unconditional means only	121.9 (120.1–123.6)			8.61			12.72
Unconditional slope model	121.0 (119.1–122.8)	2.1 (0.6–3.6)	0.005	8.67	0.27	1.00	12.41
24 h ambulatory systolic BP							
Unconditional means only	128.0 (126.3–129.8)			9.38			11.18
Unconditional slope model	127.4 (125.7–129.2)	1.7 (0.2–3.2)	0.023	10.38	2.67	−0.29	10.12
Sleep ambulatory systolic BP							
Unconditional means only	123.8 (121.9–125.8)			11.73			11.83
Unconditional slope model	123.1 (121.1–125.1)	2.3 (0.6–4.0)	0.008	12.96	3.74	−0.16	9.97
Awake ambulatory systolic BP							
Unconditional means only	130.5 (128.8–132.2)			8.23			12.04
Unconditional slope model	129.9 (128.1–131.7)	1.4 (−0.1–2.8)	0.062	9.17	1.68	−0.58	11.47

Numbers in parenthesis are the 95% confidence intervals.

decline in the PWV of 1.32 m/s in 100 patients and an increase of 0.95 m/s in 50 patients [19]. The mean change was therefore a decline of 0.85 m/s. London *et al.* reported, over a mean follow-up of 54 months of 153 hemodialysis patients, the baseline carotid-to-femoral PWV to be 11.15 m/s, which at the end of the follow-up was similar to that at baseline (11.03 m/s) [20]. However, PWV changes associate with systolic BP, which is akin to what is reported in hemodialysis patients [19, 21]. Although it is possible that BP influences the PWV or *vice versa*, it is also likely that unmeasured factors such as volume

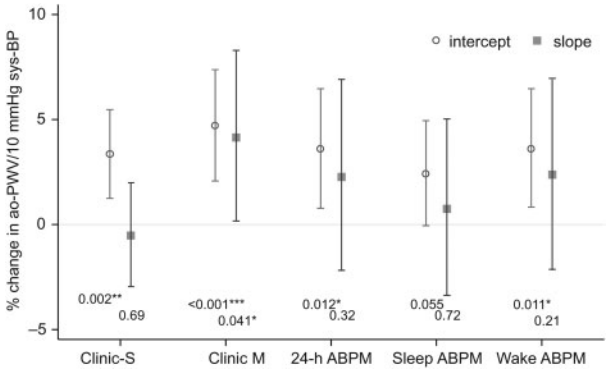

FIGURE 3: Mixed model estimates of percent change in the PWV per 10 mmHg change in systolic BP. The abscissa shows the various types of BP measurements. The intercept represents the cross-sectional association of the PWV, whereas the slope represents the longitudinal association. Multiple clinic BP measurements were superior to a single clinic BP measurement in predicting PWV growth. Awake ambulatory BP monitoring (ABPM) was superior to sleep ABPM in predicting PWV growth.

expansion may influence both outcomes: BP and PWV; thus a causal relationship should not be assumed. In contrast to the change in the PWV, longitudinal change in systolic BP was significantly different: it increased, and it did so linearly, 1.7 mmHg/year by clinic measurements and 1.8 mmHg/year by 24-h ambulatory BP measurements. Furthermore, the growth in sleep systolic BP was 60% more than that in awake ambulatory BP.

The question emerges whether it is possible to assume a relationship between the PWV and BP, one of which (PWV) did not change, on average, during the follow-up, in the whole population, and the other increased in a linear manner over time. This phenomenon can be better understood by dissecting out intra-individual change from changes in the group over time. Our data show that individuals who have an increase in the PWV also have an increase in BP. Similarly, individuals who have a fall in the PWV also have a fall in BP. Thus the two are related. At the group level, there is no change in the PWV and only a slight change in BP. However, this does not mean that the two measures do not go hand-in-hand. The random coefficient model evaluates the individual-level information and group-level information within the same model and has the power to detect the relationship at the individual level and the group level separately. Thus, the observations noted here are not inconsistent. Furthermore, the change in the PWV was related to baseline PWV; this is an important phenomenon of regression to the mean. In other words, those with a high PWV had a drop in the PWV over 2 years and those with a low PWV at baseline had a rise over 2 years. This is a phenomenon also noted for the left ventricular mass index [22].

Multiple clinic BP measurements were superior to single clinic BP measurement, taken at the time of PWV measurement, in predicting PWV velocity change. This may be because multiple clinic measurements may better reflect the intra-arterial pressure. Although a single clinic BP measurement was associated with among-participant differences in the PWV, it was unable to predict the change in the PWV over time. This suggests that carefully taken, standardized measurements of BP are particularly important for long-term studies.

Awake ambulatory BP was capable of detecting differences in the PWV among individuals but not over time. In contrast, sleep ambulatory BP was unable to discern among-individual differences in the PWV or in its longitudinal change. The author speculates that the awake and sleep BP may reflect different domains of arterial health. The awake BP is influenced by activity; in CKD we have previously reported that activity-induced changes in BP are greater in those who have been less physically active [23]. Thus, greater awake BP may reflect a more sedentary lifestyle associated with other cardiovascular risks. Variations in awake pressure through inducing pulsatile stress may stimulate more adventitial fibrosis. In contrast, sleep pressure may reflect static load and have less relevance to the progression of arterial stiffness.

The study noted that ambulatory BP was similar to well-measured oscillometric clinic systolic BP in determining the PWV at baseline and its growth over time; there were no differences in model fit. This attests to the value of well-measured clinic BP in long-term studies for the prediction of arterial stiffness. However, it should be noted that single clinic measurement was on average 14 mmHg higher than the average of three clinic visits. Still, most clinical decisions to treat or not to treat hypertension are based on a single BP measurement made at a clinic visit. Targeting BP obtained at a single clinic visit is questionable. In this study, multiple visits (three visits with each BP recording in triplicate over 1 week) were required for the detection of growth of the PWV over time. A single clinic BP measurement performed at the time of PWV assessment by Doppler ultrasound was unable to detect the growth of the PWV over time. These findings have implications for quality improvement programs and the design of interventional studies.

Limitations of the study include the following: participation in the study was restricted to veterans who are older and are predominantly men. Whether our findings apply to younger people and women will need to be clarified in future studies. Risk factors such as age, male sex, anemia, progression of kidney disease and the occurrence of cardiovascular disease may well produce PWV growth. Because all the measurements were made in the same group of participants, we did not make any adjustments simply because we could compare BP measurement techniques in a paired manner. Because all the analyses are unadjusted, we cannot conclude that BP is independently associated with PWV growth.

There are several strengths of our study: our study used 24-h ambulatory blood pressure monitoring, the gold-standard method to diagnose out-of-office hypertension. Our study was prospective; it carefully collected information on Doppler-assessed PWV solely for the purposes of the study. BP pattern assessment in each individual using several BP monitoring methods used both a single visit at the time of PWV assessment, and multiple clinic visits to define clinic hypertension, had a high completion rate.

In conclusion, among patients with CKD, nearly all of whom were taking antihypertensive medications, PWV growth is variable unlike the increase in both clinic and ambulatory BP.

Given that the growth of BP and that of PWV are concordant, attempts to control hypertension may be useful to abrogate the rate of PWV over time. BP recorded over three visits and averaged was as good as ambulatory BP monitoring and superior to a single recording of clinic BP in predicting the progression of arterial stiffness. In the usual day-to-day care of patients, it is unlikely that clinic BP will have such predictive power. Nonetheless, the study illustrates the value of well-taken clinic BP in its ability to predict between-individuals and within-individual changes in target organ damage. Differences exist with respect to the independent contributions of sleep and awake ambulatory BP on PWV growth. This may be due to the pulsatile nature of the stress that may be better captured using the awake ambulatory BP monitoring. Taken together, these data support the view that targeting clinic BP taken on multiple occasions using a standardized methodology or daytime ambulatory systolic BP may slow the progression of arterial damage.

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CONFLICT OF INTEREST STATEMENT

R.A. has been a consultant for several pharmaceutical companies that make antihypertensive drugs including Merck, Takeda, Novartis, Daiichi Sankyo, Abbvie, Bayer and Johnson and Johnson.

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